Comment on "Inferring change points in the spread of COVID-19 reveals the effectiveness of interventions"

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Abstract: Dehning et al. (Research Article, 15 May 2020: eabb9789) use inappropriate and unreliable data to show that the governmental nonpharmaceutical interventions reduced the spread of SARS-CoV-2 in Germany. Using appropriate data from official German sources, which were available for the authors, we show that just the opposite conclusion is true: The spread of the virus receded before the first governmental intervention became effective.

Main Text: In many countries, nonpharmaceutical interventions (NPI) against the spread of the coronavirus SARS-CoV-2 have been adopted that have strong negative side effects on the economy as well as on physical, mental, and social health conditions (*1-4*). Given these adverse effects, it is important to determine whether these measures were actually successful in curbing the spread of the virus. To examine this issue, Dehning et al. (5) have modelled the growth rate of SARS-CoV-2 infections in Germany using a Susceptible-Infected-Recovered (SIR) model combined with Bayesian parameter inference. The authors report change points in the growth rate that correspond closely to three NPIs that became effective on 9 March (prohibition of large public gatherings), 16 March (closing of schools and other educational institutions along with the closing of nonessential stores), and 23 March (extensive lockdown, including a contact ban). Dehning et al. conclude that the full extent of interventions was necessary to achieve a negative virus growth rate.

There are several fundamental methodological issues that cast serious doubt on the conclusions drawn by Dehning et al. Accounting for these issues suggests that the opposite of their principal inference is actually correct: neither of the governmental interventions could have had any effect on the spread of the virus because the number of new infections declined much earlier than estimated in their study. Furthermore, the authors ignore direct empirical evidence showing that such countermeasures had very low or even no effects. We consider the study by Dehning et al. (*5)* to be seriously flawed.

To assess the potential effects of NPIs on the spread of a virus, it is crucial to determine the *date of infection* as exactly as possible. With misspecified infection dates, any conclusions about the effect of NPIs are meaningless. Dehning et al. estimated the date of infection based on the date when a confirmed case was reported, according to the Johns Hopkins University Center for Systems Science and Engineering (JHU CSSE) dashboard. To infer the infection date from the reporting date, they included a parameter in their SIR model that aims at determining the so-called 'reporting delay', i.e., the delay between infection date and reporting date. Critically, their parameter estimate is constrained by an informative prior that, in turn, is based on the assumption of an incubation period of 5–6 days and a test delay. Using their priors, the authors estimated a total delay of 8.6 days during the initial phase and 11.4 days during the later phase.

This procedure is inadequate. First, Dehning et al. use data from the JHU CSSE dashboard. As the Robert Koch Institute (RKI), Germany's federal health agency, points out in its profound FAQ section on the coronavirus (*6*), data from the JHU CSSE dashboard allow only limited conclusions because they stem from internet media reports and social media, and vary in reporting guidelines. Second, inferring infection dates from reporting dates would only make sense if reporting dates varied systematically with infection dates. However, the intervals between dates of actual infections, diagnostic testing, and reporting differ vastly across people. Many suspected people were tested even before symptom onset, whereas true patients were at times tested more than 20 days after symptom onset (*7*). Therefore, it is hardly possible to conclude anything meaningful from modeling the spread of infections using reporting dates.

Germany's RKI, (*7*), published 15 April and accessible to Dehning et al. who used data up to 21 April, employs a more sophisticated approach. Their model is not based on reporting dates but on identified dates of *symptom onset*, referred to as *incident cases*. With an established incubation period of 5 days (e.g. (*8*), 5.1 days, CI 95% 4.5 to 5.8 days), incident cases reflect infection dates much more accurately. To describe the dynamics, RKI uses a growth factor R (reproduction number), which compares the 4-day mean of incident cases on one day with the corresponding mean 4 days before. By construction, R lags the actual dynamics by 4 days. To make our argument more succinct, we neglect this lag, consideration of which would strengthen our point. Fig. 1 compares the effective growth rate of *infections*, λ^*, estimated by Dehning et al. with the actual growth factor of *incident cases*, R, determined by RKI.

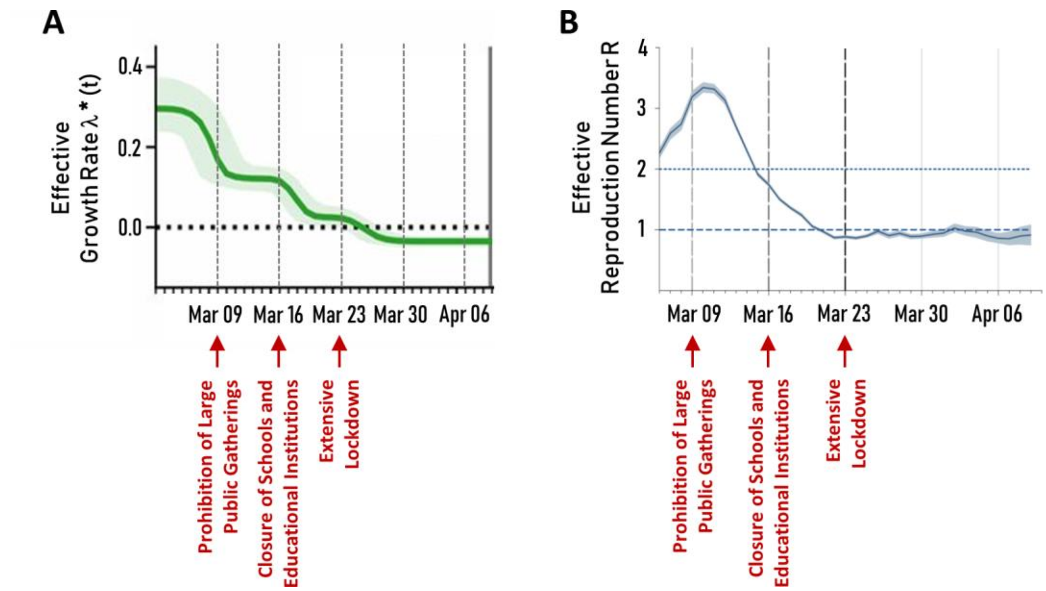

Fig. 1. Growth of infections versus growth of incident cases. **(A)** Growth rate of infections from (*5*), *Fig. 3A*. **(B)** Growth factor of incident cases from (*7*), *Fig. 4*.

Fig. 1B documents that the growth of incident cases reached its maximum already on March 10 and was negative (i.e., R < 1) since March 21. With an incubation period of 5 days, the corresponding growth of infections reached its maximum on March 5, long before the first NPI became effective, and was negative since March 16. Therefore, it is obvious that the spread of the virus was in decline before the first intervention took place, and was no longer exponential at the time of school closure and the extensive lockdown. This contradicts the main result of Dehning et al. ”that the full extent of interventions was necessary to stop exponential growth” (p. 4).

Several reasons for such an autonomous decline have been suggested. One is that differences in host susceptibility and behavior can result in herd immunity at a relatively low prevalence level. Accounting for individual variation in susceptibility or exposure to the coronavirus yields a maximum of 17% to 20% of the population that needs to be infected to reach herd immunity (*9*), an estimate that is empirically supported by the cohort of the Diamond Princess cruise ship (*10*). Another reason is that seasonality may also play an important role in dissipation (*11*). Finally, the ineffectiveness of the NPIs is also supported by empirical studies that determine their effects directly: Recent studies have shown that children are less involved in the transmission of SARS-CoV-2 than adults (*12,13*), suggesting that the closure of schools and kindergartens contributes little to curbing the spread of SARS-CoV-2. This finding is supported by studies of previous pandemics: A review of the effects of school closures regarding the spread of SARS-1 in China, Hong Kong, and Singapore in 2003 found only marginal effects (*14*).

In summary, the inferences regarding the effectiveness of nonpharmaceutical measures by Dehning et al. (*5*) are invalid for a number of reasons. Most importantly, their model implies counterfactual lags between infection dates and reporting dates. Official RKI figures suggest that the pandemic receded autonomously in Germany before any governmental measures were taken. The latter finding accords with (*12-14*). More recent results from German county data are also fully consistent with our critique (*15*).

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Acknowledgments: The authors declare that they have no conflicts of interest.